J Anesth (2009) 23:192–197
DOI 10.1007/s00540-008-0737-9

Journal of
Anesthesia

Gradient of bronchial end-tidal CO_2 during two-lung ventilation in lateral decubitus position is predictive of oxygenation disorder during subsequent one-lung ventilation

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Abstract
Purpose. Hypoxemia is one of the major problems during one-lung ventilation (OLV). During two-lung ventilation (TLV) using a double-lumen bronchial tube, bronchial end-tidal carbon dioxide partial pressure ($ETbr_{CO_2}$) can be determined on both sides, independently. The $ETbr_{CO_2}$ is mainly dependent on the pulmonary perfusion to each lung. If the degree of oxygenation disorder during OLV were to be predictable before starting OLV, this could provide time to prepare for any subsequent hypoxemia. The aim of this study was to investigate whether the difference of $ETbr_{CO_2}$ (D-$ETbr_{CO_2}$) between the dependent and the nondependent lungs during TLV in the lateral decubitus position (LP) could be a predictive factor for the severity of oxygenation disorder under subsequent OLV.
Methods. Eighteen patients undergoing lung surgery were enrolled in this study. Anesthesia was induced with intravenous thiopental and fentanyl, supplemented by the inhalation of sevoflurane. A left-sided double-lumen bronchial tube was placed. The $ETbr_{CO_2}$ was independently determined on each side during TLV in the supine position (SP) and at 10 min after changing the position from SP to LP. Pa_{O_2}/ inspiratory fraction of oxygen ($F_{I_{O_2}}$) was taken at 15 min after switching from TLV to OLV in LP.
Results. The decrease of $Pa_{O_2}/F_{I_{O_2}}$ at 15 min during OLV in LP correlated with the reduction of the D-$ETbr_{CO_2}$ predetermined during TLV in LP ($r = 0.698$; $P < 0.01$).
Conclusion. The D-$ETbr_{CO_2}$ predetermined during TLV in LP could be a predictive factor for the severity of oxygenation disorder after starting OLV in LP.

Key words One-lung ventilation · Hypoxemia · Bronchial end-tidal ventilation · Lateral decubitus position

Introduction

Despite advances in surgical techniques and therapeutic options, hypoxemia remains one of the major problems during one-lung ventilation (OLV) in the lateral decubitus position (LP). The misplacement of a bronchial tube, the development of atelectasis in the dependent lung, and the inhibition of hypoxic pulmonary vasoconstriction (HPV) in the nondependent lung have been recognized to be common factors causing oxygenation disorder during OLV [1,2]. The arterial blood oxygen partial pressure (Pa_{O_2}) during OLV in LP is influenced by the ventilation/perfusion (V/Q) ratio, especially the amount of shunting pulmonary blood flow in the nondependent lung. On the other hand, the pulmonary blood perfusion of each lung during OLV in LP depends on gravitational force, HPV, and preoperative pulmonary blood distribution.

During two-lung ventilation (TLV) using a double-lumen bronchial tube, bronchial end-tidal carbon dioxide partial pressure ($ETbr_{CO_2}$) and the tidal volume of each lung (TVbr) can be independently determined on each side. Because the end-tidal carbon dioxide pressure (ET_{CO_2}) is mostly influenced by the pulmonary blood flow [3,4], each lung $ETbr_{CO_2}$ reflects the gross pulmonary blood distribution between the dependent and nondependent lungs. The preoperative state of perfusion in each lung was reported to be one of the predictors of hypoxemia in OLV [5–8]. The online determination of each lung $ETbr_{CO_2}$ prior to starting OLV is easier, time-saving, and noninvasive compared with a visualizing examination of lung perfusion using a radioisotope.

If the severity of oxygenation disorder during OLV could be predictable before starting OLV, this could provide time to spare for coping with the hypoxemia after switching to OLV. This clinical study was conducted to investigate whether the difference in the $ETbr_{CO_2}$ (D-$ETbr_{CO_2}$) between the dependent and non-

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Received: October 28, 2008 / Accepted: December 25, 2008

dependent lungs during TLV in LP could be a predictable factor for the occurrence of oxygenation disorder during subsequent OLV in LP.

Subjects, materials, and methods

This clinical study was performed after approval of the protocol by the University Ethics Committee. Informed consent was obtained from all of the participants before starting the study. The participants consisted of 18 adult patients who underwent open-chest surgery in LP for lung cancer. Patients with severe chronic obstructive pulmonary disease (American Society of Anesthesiologists [ASA] physical status 3–5) or those who had a recent history of bronchial asthma attacks were excluded from this study. Patients with apparent laterality or severe adhesion, due to old tuberculosis, on chest radiographs were also excluded. All patients were premedicated with intramuscular midazolam (2 mg) 30 min before arrival at the operating room. The monitoring of electrocardiograms, pulse oxymetry, and blood pressure was started. An intravenous catheter was then placed on the forearm and acetate acid Ringer's solution was administered at 200 to 300 $ml \cdot h^{-1}$. An epidural catheter was introduced for postoperative pain management at the T4–5 or T5–6 vertebral level, with the patient in the left LP before the induction of anesthesia, and no drug, except for 2 ml 2% mepivacaine as a test dose, was injected through the catheter until the study was completed.

Anesthesia was induced with intravenous thiopental ($5\ mg \cdot kg^{-1}$) combined with fentanyl (0.1 mg), supplemented by the inhalation of 1%-2% sevoflurane in oxygen. A left-sided double-lumen endobronchial tube (35–37 Fr; Broncho-Cath; Mallinckrodt, Athlone, Ireland) was placed after the administration of intravenous vecuronium ($0.1\ mg \cdot kg^{-1}$). The endobronchial tube was properly placed under bronchofiberscopy. The proper placement of the tube was reconfirmed after body positioning from SP to LP and also at the end of surgery. The tidal volume (TV) was set at $10\ ml \cdot kg^{-1}$ with the volume preset mode and respiratory rate at 12 $breaths \cdot min^{-1}$. The ventilatory setting was never varied throughout the study, even during the period of OLV in LP. The radial artery was cannulated for blood pressure monitoring and arterial blood gas analysis. Subsequent anesthesia was maintained with incremental doses of additional intravenous fentanyl (total 0.2 mg) and inhalation of sevoflurane in oxygen mixed with air with a total flow of $4\ l \cdot min^{-1}$ as needed. The esophageal temperature was maintained at around 36.5°C throughout the study.

The inspiratory fraction of oxygen ($F_{I_{O_2}}$) and TV were determined at a site proximal to the merging portion of the two bronchial tubes by a monitor equipped with a ventilator in the respiratory circuit (Datex-Ohmeda 7900 Ventilator; Datex Ohmeda, Madison, WI, USA; Fig. 1). The ET_{CO_2} was also taken at a site proximal to the merging portion by interposing a flow-through type instrument (Viridia CMS2000; Philips, Boeblingen, Germany). At the same time, the $ETbr_{CO_2}$ and TVbr were also independently determined on each site distal to the merging portion of bronchial tubes by interposing another two gas-sampling type instruments ($200\ ml \cdot min^{-1}$; Capnomac Ultima; Datex-Ohmeda, Helsinki, Finland). The monitoring adapters were interposed without remodeling the double-lumen endobronchial tube. The D-$ETbr_{CO_2}$ was calculated by subtracting the $ETbr_{CO_2}$ in the nondependent lung from the $ETbr_{CO_2}$ in the dependent lung. Measurements were repeated at the following six points while patients were under general anesthesia, with the placement of a double-lumen endobronchial tube: in SP during TLV; during TLV at 10 min after positioning to LP; and 0, 5, 10, and 15 min after starting OLV in LP. Switching from

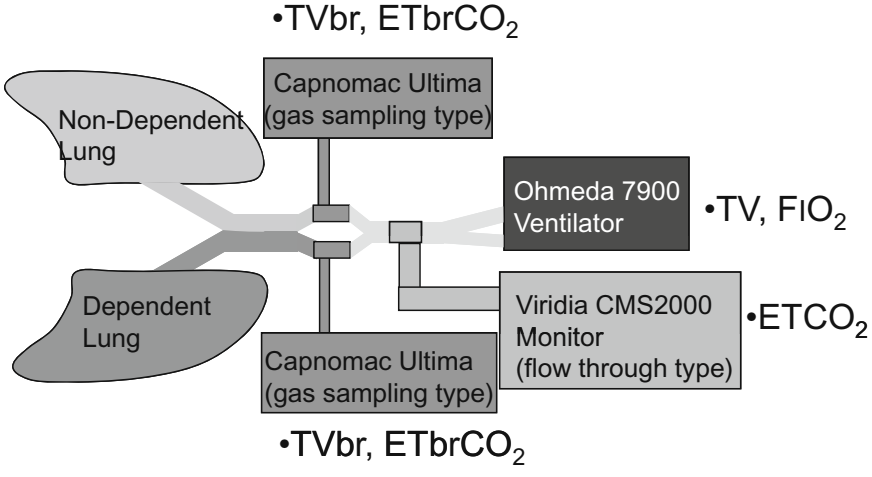

Fig. 1. Schematic drawing of the sites and instruments for the measurements of tidal volume (*TV*), inspiratory fraction of oxygen ($F_{I_{O_2}}$), end-tidal carbon dioxide pressure (ET_{CO_2}), tidal volume of each lung (*TVbr*), and bronchial end-tidal carbon dioxide pressure ($ETbr_{CO_2}$)

TLV to OLV was performed immediately before surgical opening of the chest wall.

Data analysis was conducted using a statistical software package (SPSS Ver. 11.5J; SPSS, Chicago, IL, USA). The sequential changes of each variable over time were compared using repeated analysis of variance. Furthermore, the values were compared using post-hoc tests with Bonferroni's correction. Changes in the $ETbr_{CO_2}$ and TVbr levels from SP to LP during TLV were compared by the paired *t*-test. The relationship between the values of D-$ETbr_{CO_2}$ and $Pa_{O_2}/F_{I_{O_2}}$ was tested by linear regression analysis. Differences were considered to be significant at $P < 0.05$. All data values were expressed as means ± SD.

Results

The baseline characteristics of the patients, including the preoperative results of arterial blood gas analysis and spirograms, are presented in Table 1. Both the arterial blood pressure and the heart rate were maintained at a constant level from SP to LP during TLV (Table 2).

Although Pa_{O_2} and $Pa_{O_2}/F_{I_{O_2}}$ did not change during TLV after changing the body position from SP to LP, they gradually decreased as the time after the start of OLV was extended in LP (Table 3). Pa_{O_2} and $Pa_{O_2}/F_{I_{O_2}}$ demonstrated no differences between 10 min and 15 min after switching from TLV to OLV in LP. In the right and left decubitus positions, Pa_{O_2} during OLV was 224 ± 23 mmHg and 136 ± 23 mmHg, respectively ($P < 0.01$). On the other hand, Pa_{CO_2} was maintained at around 35 mmHg with keeping of the initially set TV, even after the switching to OLV from TLV in LP.

The TV remained almost constant in LP during OLV as well as during TLV (Table 4). However, TVbr in the nondependent lung increased from 279 ± 57 ml (49 ± 5 % of TV) to 322 ± 63 ml (55 ± 5 %) after positioning the patients from SP to LP during TLV. Conversely, TVbr in the dependent lung decreased from 284 ± 51 ml (51 ± 5 % of TV) to 254 ± 44 ml (44 ± 5 %).

The total ET_{CO_2} remained almost constant throughout the study, except immediately after starting OLV in LP (Table 5). The mean $ETbr_{CO_2}$ increased in the dependent lung from 33 mmHg to 36 mmHg ($P < 0.01$) and decreased in the nondependent lung from 33 mmHg to 29 mmHg ($P < 0.05$) at 10 min after positioning the patients from SP to LP during TLV. The $ETbr_{CO_2}$ in the dependent lung decreased only at the beginning of OLV and returned to the level seen during TLV in SP.

There was a significant correlation between the $Pa_{O_2}/F_{I_{O_2}}$ at 15 min after starting OLV in LP and the predetermined D-$ETbr_{CO_2}$ during TLV in LP ($P < 0.01$; $r = 0.698$; Fig. 2). Other factors, including age, sex, and preoperative blood gas and respiratory function tests did not show any correlation with $Pa_{O_2}/F_{I_{O_2}}$ during OLV in LP.

Table 1. Baseline characteristics of the 18 patients

Sex (F/M)	10/8
Age (years)	66 ± 9
Body weight (kg)	54 ± 10
Body height (cm)	156 ± 9
Arterial blood gas (room air)	
pH	7.42 ± 0.04
Pa_{O_2} (mmHg)	93 ± 15
Pa_{CO_2} (mmHg)	41 ± 5
Spirogram	
Vital capacity (l)	3.12 ± 0.65
(%)	109 ± 17
FEV 1.0 (l)	2.19 ± 0.74
(%)	72 ± 12
Surgical position	
r-LP	7
l-LP	11
Lung surgery performed	
Partial resection	7
Lobectomy	10
Pneumonectomy	1

Values are means ± SD
FEV 1.0, forced expiratory volume in 1 s; r-LP, right lateral decubitus position; l-LP, left lateral decubitus position

Table 2. Sequential changes in arterial pressure and heart rate before and after starting one-lung ventilation

Ventilation	Body position	Time	Systolic AP (mmHg)	Diastolic AP (mmHg)	Heart rate (beats·min^{-1})
TLV	SP		107 ± 20	60 ± 13	64 ± 9
TLV	LP	10 min	103 ± 14	60 ± 8	59 ± 7
OLV	LP	0 min	128 ± 24[2]*	74 ± 14[2]*	64 ± 9
OLV	LP	5 min	114 ± 23	66 ± 14	65 ± 8
OLV	LP	1 min	118 ± 26[1]*	69 ± 15	69 ± 10[4]*
OLV	LP	15 min	112 ± 21	64 ± 11[3]*	68 ± 10[4]*

[1]* $P < 0.05$ vs during TLV in LP; [2]* $P < 0.01$ vs during TLV in LP; [3]* $P < 0.05$ vs 0 min during OLV in LP; [4]* $P < 0.01$ vs during TLV in LP
Data values are presented as means ± SD
AP, arterial pressure; TLV, two-lung ventilation; OLV, one-lung ventilation;, SP, supine; LP, lateral decubitus position; OLV LP 10 min, 10 min after positioning from SP to LP; OLV LP 0 min, 5 min, 10 min, 15 min, time after switching from TLV to OLV in LP

Table 3. Sequential changes in arterial blood gas variables before and after starting one-lung ventilation

Ventilation	Body position	Time	$F_{I_{O_2}}$	pH	Pa_{O_2} (mmHg)	$Pa_{O_2}/F_{I_{O_2}}$	Pa_{CO_2} (mmHg)
TLV	SP		0.90 ± 0.18	7.44 ± 0.05	496 ± 98	550 ± 49	35 ± 5
TLV	LP	10 min	0.85 ± 0.21	7.43 ± 0.04	445 ± 123	522 ± 51	35 ± 5
OLV	LP	0 min	0.63 ± 0.11	7.43 ± 0.04	272 ± 74[1*;2*]	439 ± 119[1*]	34 ± 5
OLV	LP	5 min	0.64 ± 0.10	7.43 ± 0.03	153 ± 71[1*;2*;3*]	250 ± 127[1*;2*;3*]	35 ± 4
OLV	LP	10 min	0.67 ± 0.14	7.44 ± 0.03	122 ± 40[1*;2*;3*]	191 ± 79[1*;2*;3*;4*]	34 ± 4
OLV	LP	15 min	0.69 ± 0.14	7.44 ± 0.04	120 ± 37[1*;2*;3*]	182 ± 69[1*;2*;3*;4*]	34 ± 3

Data values are presented as means ± SD
$F_{I_{O_2}}$, inspiratory fraction of oxygen; TLV, two-lung ventilation; OLV, one-lung ventilation; SP, supine; LP, lateral decubitus position; TLV LP 10 min, 10 min after positioning from SP to LP; OLV LP 0 min, 5 min, 10 min, 15 min, time after switching from TLV to OLV in LP
[1*] $P < 0.01$ vs during TLV in SP; [2*] $P < 0.01$ vs 10 min during TLV in LP; [3*] $P < 0.01$ vs 0 min during OLV in LP; [4*] $P < 0.05$ vs 5 min during OLV in LP

Table 4. Sequential changes in actual tidal volume determined before and after starting one-lung ventilation

				TVbr (nondependent lung)		TVbr (dependent lung)	
Ventilation	Body position	Time	TV (ml)	(ml)	(%)	(ml)	(%)
TLV	SP		533 ± 82	279 ± 57	49 ± 5	284 ± 51	51 ± 5
TLV	LP	10 min	529 ± 71	322 ± 63**	55 ± 5**	254 ± 44*	44 ± 5**
OLV	LP	0 min	505 ± 56	NA	NA	561 ± 79	NA
OLV	LP	5 min	497 ± 50	NA	NA	553 ± 74	NA
OLV	LP	10 min	491 ± 51	NA	NA	551 ± 66	NA
OLV	LP	15 min	497 ± 58	NA	NA	547 ± 75	NA

* $P < 0.05$ vs during TLV in SP; ** $P < 0.01$ vs during TLV in SP, by paired *t*-test
Data values are presented as means ± SD
TV showed no significant differences by repeated analysis of variance
As the TV and TVbr were determined at separate sites, the sum of TVbr in each lung did not always accord with TV
NA, not available; TV, tidal volume; TVbr, tidal volume of each lung; TLV, two-lung ventilation; OLV, one-lung ventilation; SP, supine; LP, lateral decubitus position; TLV LP 10 min, 10 min after positioning from SP to LP; OLV LP 0 min, 5 min, 10 min, 15 min, time after switching from TLV to OLV in LP

Table 5. Sequential changes in end-tidal CO_2 before and after starting one-lung ventilation

Ventilation	Body position	Time	ET_{CO_2} (mmHg)	$ETbr_{CO_2}$ (nondependent lung) (mmHg)	$ETbr_{CO_2}$ (dependent lung) (mmHg)
TLV	SP		30 ± 5	33 ± 5	33 ± 5
TLV	LP	10 min	29 ± 4	29 ± 5[1*]	36 ± 5[2*]
OLV	LP	0 min	27 ± 4	NA	30 ± 4[4*]
OLV	LP	5 min	29 ± 4[5*]	NA	32 ± 4[4*;6*]
OLV	LP	10 min	29 ± 3[5*]	NA	33 ± 4[3*;6*]
OLV	LP	15 min	29 ± 3	NA	32 ± 3[4*;5*]

[1*] $P < 0.05$ vs during TLV in SP, [2*] $P < 0.01$ vs during TLV in SP by paired *t*-test; [3*] $P < 0.05$ vs during TLV in LP; [4*] $P < 0.01$ vs 0 min during OLV in LP, by multiple comparison with Bonferroni's correction; [5*] $P < 0.05$ vs 0 min during OLV in LP; [6*] $P < 0.01$ vs 0 min during OLV in LP, by multiple comparison with Bonferroni's correction
Data values are presented as means ± SD
NA, not available; ET_{CO_2}, tracheal end-tidal carbon dioxide; $ETbr_{CO_2}$, bronchial end-tidal carbon dioxide; TLV, two-lung ventilation; OLV, one-lung ventilation; SP, supine; LP, lateral decubitus position; TLV LP 10 min, 10 min after positioning from SP to LP; OLV LP 0 min, 5 min, 10 min, 15 min, time after switching from TLV to OLV in LP

Discussion

We found a significant correlation between the $Pa_{O_2}/F_{I_{O_2}}$ at 15 min after starting OLV in LP and the D-$ETbr_{CO_2}$ predetermined during TLV in LP ($P < 0.01$; $r = 0.698$). The D-$ETbr_{CO_2}$ was obtained by subtracting the $ETbr_{CO_2}$ in the nondependent lung from the $ETbr_{CO_2}$ in the dependent lung. Because ET_{CO_2} is influenced by pulmonary perfusion [3, 4], the D-$ETbr_{CO_2}$ could be affected by the distribution of perfusion between the dependent and nondependent lungs. The reduction of $Pa_{O_2}/F_{I_{O_2}}$ during OLV would also be affected by the distribution

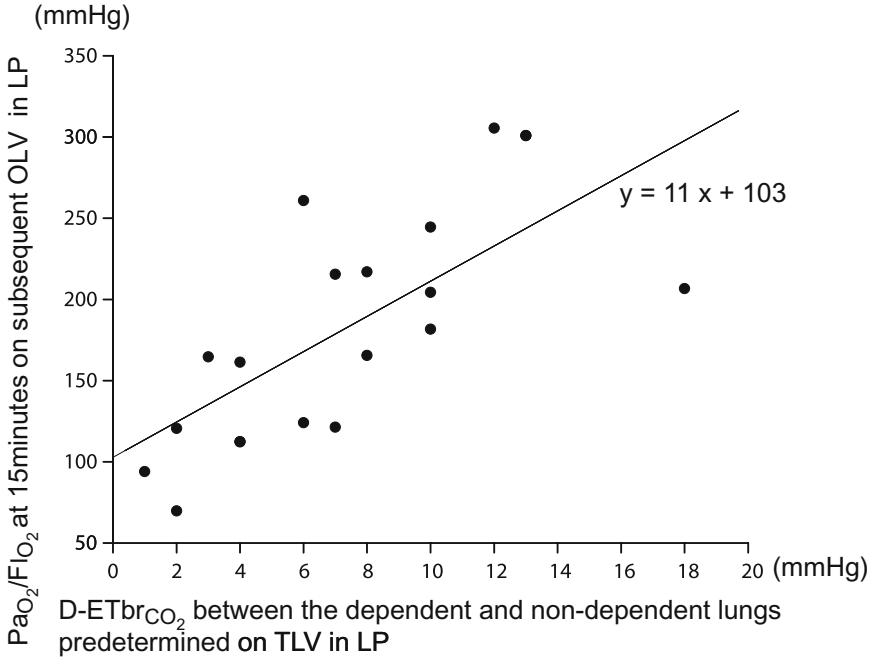

Fig. 2. Relationship between the difference in the bronchial end-tidal carbon dioxide pressure between the nondependent and dependent lungs (*D-ETbr*$_{CO_2}$) predetermined during two-lung ventilation (*TLV*) in the lateral decubitus position (*LP*) and reduction of $Pa_{O_2}/F_{I_{O_2}}$ during subsequent one-lung ventilation (*OLV*) in LP. As the D-$ETbr_{CO_2}$ during TLV in LP decreased, $Pa_{O_2}/F_{I_{O_2}}$ at 15 min during subsequent OLV in LP showed lower values ($P < 0.01$; $r = 0.698$)

of lung perfusion, especially by the amount of shunt flow in the nondependent lung. If the D-$ETbr_{CO_2}$ represents the difference of pulmonary blood flow between the dependent and nondependent lungs, then our results are coincident with those of previous studies [5–8], in which the distribution of pulmonary blood flow was preoperatively determined by using a radioisotope. Clinically, the D-$ETbr_{CO_2}$ can be obtained instantly and noninvasively by interposing a pair of measurement instruments in the respiratory circuit. In fact, two instruments are not always necessary when the $ETbr_{CO_2}$ is determined at each site in turn. The early determination of the D-$ETbr_{CO_2}$ during TLV in LP allows us time to prepare for high-frequency jet ventilation and continuous positive airway pressure in the nondependent lung [9].

There are some limitations of our study. The $ETbr_{CO_2}$ will be influenced not only by the pulmonary blood flow but also by the ventilatory volume and the dead space. Furthermore, the expiratory CO_2 determination method can detect only 60 % of actual pulmonary flow reduction compared with the electromagnetic flowmeter method, in which the flow probe is fitted directly to the pulmonary artery, and SF6, a poorly soluble gas, is used [4]. Because CO_2 is more soluble in the blood than SF6, the expiratory CO_2 determination method is more suitable for the detection of V/Q mismatching.

In addition to the preoperative distribution, the pulmonary blood flow during OLV is influenced by various factors. Opening of the chest wall during OLV causes the collapse and decreased volume of the nondependent lung. The lung volume was reported to be one of the factors in the distribution of pulmonary blood flow. Simmons et al. [10], in an animal study, showed that the pulmonary vascular resistance increased when the lung was diminished in volume below the functional residual capacity. This finding has not been confirmed in humans. Another factor affecting pulmonary shunt is hypoxic pulmonary vasoconstriction (HPV). HPV was shown to appear within a few seconds after the reduction of $F_{I_{O_2}}$ in an animal study [11]. Morrell et al. [12] also observed, in humans, that hypoxia in a single lobe caused a rapid decline in the perfusion of the lobe. After 5 min, the pulmonary perfusion of the hypoxic lobe was reduced to half. It is possible that, in our study, HPV in the nondependent lung could have occurred after starting OLV in LP; however, we did not conduct alveolar gas analysis in the nondependent lung during OLV. Gravitational force is assumed to have affected the pulmonary perfusion, not only during TLV but also during OLV. The amount of pulmonary perfusion in LP is larger in the dependent lung than in the nondependent lung. Therefore Pa_{O_2} during OLV is kept comparatively higher in LP than in SP [13].

The D-$ETbr_{CO_2}$ was also influenced not only by the preoperative distribution of pulmonary blood flow but also by the effect of gravitational force. By changing the body position from SP to LP during TLV, the $ETbr_{CO_2}$ was decreased in the nondependent lung and increased in the dependent lung. Pulmonary perfusion is known to be distributed proportionally to the gradient of gravitational force. When the body position of patients is changed from SP to LP, pulmonary perfusion shifts from the nondependent lung to the dependent lung.

When inadequate differential two-lung ventilation occurs because of misplacement of a double-lumen endobronchial tube or cuff leakage, $ETbr_{CO_2}$ represents the double-peak waveform. In the present study, the value of ET_{CO_2} was always lower at the tracheal level than at the bronchial level at all times of determination. The difference in the expiratory CO_2 pressures may be affected, in part, by the difference in the dead space between the determination sites for ET_{CO_2} and $ETbr_{CO_2}$.

Of interest, the change in body position in our study caused an increase of the TVbr in the nondependent lung and a decrease of that in the dependent lung. Wulff and Aulin [14] previously indicated that, during TLV in an anesthetized patient in LP, the preferential distribution of tidal volume to the nondependent lung was enhanced. The reason that the sum of each TVbr in our study was not identical with the TV was that each TV was obtained at three different sites in the respiratory circuit.

There have been several reports describing the predictors of hypoxemia during OLV in LP. Hurford et al. [5] suggested preoperative lung scanning denoting the distributions of perfusion and ventilation of the operative lung as a predictor. Ribas et al. [6] used Pa_{O_2} during exercise, preoperative perfusion of the dependent lung, and the LP side as predictors. The influence of positional laterality in our study is similar to the findings of previous reports [6,15]. Nomoto [7] suggested the distribution of preoperative lung perfusion and the percentage of predicted vital capacity as predictors of hypoxemia. Slinger et al. [15] indicated that the LP side, the preoperative percent forced expiratory volume in 1 s and the intraoperative Pa_{O_2} during TLV may be predictors of hypoxemia. Guenoun et al. [8] showed several factors to be predictors of hypoxemia, such as age, hematocrit level, relative perfusion to the operative lung, intraoperative Pa_{O_2} during TLV, and mean arterial pressure at the lowest Pa_{O_2} during OLV. The prediction of Pa_{O_2} during OLV still implies that several problems remain to be solved, because it includes both preoperative and intraoperative factors.

In summary, we found that the $D\text{-}ETbr_{CO_2}$ during TLV in LP correlated with the Pa_{O_2}/FI_{O_2} reduction during subsequent OLV in LP. The early determination of $D\text{-}ETbr_{CO_2}$ during TLV in LP allows us time to prepare for an oxygenation disorder during the following OLV in LP.

Acknowledgments. This work was supported by a Grant-in-Aid from the Japanese Ministry of Education, Culture, Sports, Science and Technology for Young Scientists (B, 19791093).

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